something. But when the ulcer is infected, it's painful, it's red, it's angry looking. In those individuals we have found that a course of IV antibiotics will quickly control the infection, and then you can go to further measures.

DR. BELCARO: Just an assumption. Every ulcer is infected. According to cultures, you will always find something there. So almost by definition as long as you have the skin open, there is an infection. So I think you should use antibiotic treatment any time you have an open ulceration.

UNIDENTIFIED SPEAKER: Can I come back on that? That's absolutely right. Even if you just deal with antibiotics, you're not going to get rid of all these parts. What you need to do, once you actually aim to heal it with skin graft, is just to actually protect your skin graft for the appropriate organism. It doesn't matter if you've got pseudomonas, staph, whatever. Skin grafts will take providing there is a good bed. What you need is to protect it from a hemolytic strep.

DR. PERRIN: I would propose that the patients would be assessed one month after surgery by a duplex scan in order to identify any perforating vein which was missed, or a collateral branch, in order to treat them because it is easy to miss something in this kind of patient. I think duplex is very helpful.

DR. TRIPATHI: Based on the experience with diabetic ulcers, what we in our center do with the venous ulcers is to take a superficial scrape and also use aspiration culture from the deeper layers of the venous ulcer. If the deeper layer culture is negative, then we go ahead and we scrape the superficial layer and put skin grafts in the OR with prophylactic antibiotics. I have never had a problem. I don't know if other people have experienced the same.

DR. PARSI: I'm interested in the role of microthrombi in the pathophysiology of this sort of ulcer. As you know, a lot of these patients have thrombophilia. Up to 26 percent of patients with ulcers have Activated Protein. Up to 40 percent have anticardiolipin C Resistance antibodies. Were these patients screened at all? Some of the thrombophilias like MTHR mutation with high homocysteine levels, can actually be treated with folic acid.

DR. O'DONNELL: That's a very good point. Certainly with deep venous reconstruction in our unit or in patients undergoing SEPS we would do a screening. What about this lady? Would most of you do a hematologic coagulation screen?

DR. KISTNER: No. This is pure primary reflux disease. I don't think she has any sign of thrombotic disease.

DR. DEPEDRO: A small comment. Permanent venous hypertension in this patient, which of the members of the panel think that this kind of ulceration is due to the insufficiency alone of the long saphenous vein and which think that this is due to the atrophy of the muscular pump?

DEPALMA: I think it's due to both factors, and I think she's absolutely right. There is wasting of the calf muscle. My idea in operating on it rapidly is to get it covered so that the patient can move the ankle around.

DR. RAJU: I think these massive ulcers are larger than what you think would be appropriate for isolate saphenous reflux. That seems to be somewhat more common in the older age group. We have seen a number of seventies and eighties with this kind of presentation that you don't see in the younger age group. So sclerosis of the deep veins and compliance changes might have something to do with it. I wonder whether anybody in the panel has seen the younger patient with this kind of isolated saphenous reflux with massive ulceration.

DR. KISTNER: The patient was 65 years old according to the history and had her first ulcer at 30 years. It brings up the question of where this patient has been in the 30 years. Has she been under any therapy at all or just totally neglected? The reason to raise this point is that there exists a mass of medical practitioners who have no understanding of this whole problem, and they would treat with a salve but never provide any compression. I wonder if this patient fits into that category.

SURGICAL MANAGEMENT

DR. EKLOF: This is an unusual case to show for saphenous vein incompetence. I took this case because I think it's a badly treated case for 30 years. The only alternative she was offered before was amputation of the leg, indicating how far we have to go to get widespread understanding of how to deal with sick patients like this. She had poor treatment until the most senior of surgeons in Hawaii, the first surgeon who got his American Board in Surgery sent her over to me, Lester Yee. He is now in his eighties and still practicing. I think he saved her at least from amputation. We admitted the patient, treated the infection, and cleaned up the ulcer. The reason we did the venograms was that I thought that this was more than just saphenous vein incompetence. It was such a longstanding ulceration. We couldn't find any perforators with a scanner under the ulcer. She had no perforators above the ulcer. We did high ligation and stripping of the GSV to just above the ulceration, and skin grafted the ulcer, which healed in about a week (Fig. 3). This was about three years ago. I tried many times to scan her, and I offered her to come to the hotel tomorrow to be part of the workshop, and scan her leg, but she could not make it. So I don't know more about the perforators in this patient. Pathology of the vein that we removed showed an arterialized vein due to the long standing turbulent reflux for many years.

II. PERFORATOR INCOMPETENCE

CASE OF PERFORATOR INCOMPETENCE - MAKING SEPS BETTER

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Perforator vein incompetence contributes to ulceration when abnormally elevated pressure is transmitted to the skin usually at the ankle medially. To correct this, a variety of surgical techniques have evolved; I suggest technical modifications of SubFascial Endoscopic Surgery (SEPS) to include extrafascial submalleolar perforator division and combinations of other interventions.

In 1966, Linton's approach to perforators was modified by eliminating longitudinal incisions, creating a series of bipedicle flaps in natural skin lines and avoiding areas of severe skin involvement. This procedure was performed by remote subcutaneous access

obliterating perforating veins from the crest of the tibia to the submalleolar region. Long-term results with this approach were reported in 1974.[1] No major wound infection or necrosis of flaps occurred. The ligation of inframalleolar and foot perforators as well as those in the calf was emphasized. The ulcer was dissected subfascially and perforators in the ulcer bed ligated directly and skin grafts applied in one operation. Using this approach, the recurrence rate for venous ulcers ranged from 9 to 10% in 168 limbs amongst 141 patients with observations extending 5 to 10 years in three series.[3,4,5] We later used a phlebotome passed subcutaneously in a line to interrupt the posterior venous arcade and Cockett's perforators.[6]

With the availability of Duplex scanning, and the realization that a more precisely focused approach could alter transmission of venous hypertension to the skin, the scene was set for less invasive procedures. At the same time, endoscopic techniques were developed resulting in specialized equipment. In 1985, Hauer reported an experience using endoscopic techniques to divide perforating veins in the subfascial space in the lower extremity.[7] Two retrospective series from Europe, Jugenheimer, et al 1992[8] and Pierik, et al 1995,[9] described their experience with SEPS. Variations of the technique were subsequently described in the literature.[10,11,12,13] In 1997 the North American SEPS Registry results were presented at the Society for Vascular Surgery, reporting on 148 patients from 17 different centers. Although the results as reported in the Registry, the largest study to date, continued to be encouraging, a disappointing 22% ulcer recurrence rate at 30 months,[15] suggested a need to re-evaluate not only indications for SEPS but technical modifications which might improve results.

Indications

This procedure is used for patients with severe CVI clinical disease, CEAP Class 4 to 6.[16] It is used most frequently in our practice for Class 5 or 6 patients, who, by definition, have active or immediately past ulceration. The procedure is employed for patients with previous deep venous thrombosis, valvular incompetence, or combined abnormalities, which may be corrected whenever practical. Patients with reflux tend to have better outcomes. Unless correctable, we consider caval occlusion to be a contraindication to SEPS as a sole procedure.

Preoperative evaluation

Duplex scanning includes examination of the greater saphenous veins and deep veins for obstruction and valvular incompetence, as well as identification of incompetent perforator veins. It is important that this be done in a standing position. We now recommend ascending and descending phlebography for all patients, in addition to duplex scanning. These procedures are best for finding areas of recanalized thrombosis or to locate incompetent perforators.[17] Phlebography is needed to plan combined operations such as valvular repair, inframalleolar perforator interruption, and iliac bypass for occlusive disease.

Surgical Technique

SEPS is a relatively new procedure and techniques vary somewhat. We no longer use a tourniquet or exsanguination and we use two port systems placed just below the knee. When two ports are used, incision placement is crucial to prevent dueling instruments. We use a balloon dissector[11] inserted through an initial transverse incision in the fascia with saline inflation to expand the balloon's width and length, thus creating an elongated bloodless working space. The balloon is then removed and replaced by an endoscopic probe which provides a constant flow of carbon dioxide to maintain expansion of the subfascial space. This port also has an endoscope and light source, which are introduced into the created space at this point. A working port is then placed under direct visualization. Perforating veins ranging in number from 6 to 8, involving the Cockett 2 and 3 complexes are divided through this working port.

When inframalleolar perforators are present, i.e. Cockett 1, which is located on average 7 cm above the calcaneous, we have used two approaches. A 14 gauge mini-port has been inserted and the subfascial ligation of this perforator is carried out. On the other hand, when an ulcer is present which requires skin grafting, the ulcer is excised and extrafascial technique is applied in the lower third of the leg and inferior to the malleolus to include the foot perforators.

Results

Twenty-six Class 5-6 patients have received operative interventions. All patients received Duplex scanning along with ascending and descending phlebography. Depending on the patterns of reflux or obstruction, differing interventions were used, including: superficial stripping 18; perforator interruptions 26: 12 extrafascial, 14 SEPS; valveplasty 2; and Palma cross over 2. Twenty three extremities remain healed at follow up, ranging from 15 to 50 months; three ulcers recurred, two healed promptly after a second operation either using SEPS or extrafascial interruption of the lowest Cockett or foot perforators. One recurrent ulceration persists; this patient declined further intervention.

Discussion

The largest study to date, the North American Subfascial Endoscopic Perforator Surgery Registry Report,[14] provided data from 17 medical centers including ours, between June 1993 and February 1996. The preliminary report looked at safety, feasibility and early efficacy of the procedure and was a retrospective analysis of 146 patients undergoing SEPS with different surgeons, medical centers, techniques and instrumentation. Mean follow up was 5.4 months and an ulcer healing rate of 88% was reported. There was evidence of more rapid ulcer healing as compared to non-operative treatment. No postoperative deaths or early thromboembolism occurred. Since that time, at our center, we have experienced a single episode of postoperative thromboembolism which was effectively treated with anticoagulation.

Clinical scores using CEAP grading have shown improvements ranging from 9.4 to 2.9 after surgery. A further study of intermediate results was published in 1999.[15] Although early results were encouraging, the 22% ulcer recurrence rate at 30 months was not. Recurrence is most common in CVI due to obstruction rather than valvular incompetence. I believe that in order to improve the results of SEPS, increased attention to submalleolar and foot perforators will be required. The subfascial space at the ankle at the malleolus is quite tight and difficult to access. While we have used SEPS approach with a separate 14 gauge port in the lower leg. I believe it is best to deal with these perforators using an extra-fascial approach.

These can be documented by ascending phlebography and can also be located immediately pre-operatively using a Doppler technique. As Kistner[17] recommended, one should not hesitate to use combinations of procedures beginning with the simplest as determined by preoperative duplex scanning, and ascending and descending phlebography. Varying interventions in the deep system might be needed; particularly caval or iliac obstructions which can be missed by limb duplex scanning. These respond poorly to perforator interruption. SEPS is a useful and elegant procedure. Results can probably be improved by additional interventions. Among these, extra-fascial ablation of the submalleolar perforators is advisable when these contribute to skin changes.

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THE VENOUS ULCER

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The venous ulcer is the most important epiphenomena occurring in the course of the Chronic Venous Insufficiency (C.V.I.) Syndrome. When present, its onset strongly affects the patient, who is unable to continue his normal activity. In our experience, from 444 cases followed up for 10 years, 50.67% presented an ulcer during this lapse. This percentage rose in posthrombotic syndromes (65.51%) and decreased in those patients who did not present deep venous pathology (46.25%).

As aforementioned, the venous ulcer is a further episode in the course of indurative hypodermitis of the C.V.I. Syndrome, and therefore its pathophysiology is closely related. This disease is a product of the permanent reflux towards the superficial system during muscular contractions, through the insufficient Direct Perforant Venous System.

In our series, patients were evaluated with phlebography and phlebomanometry. Among them, 56,53% had a history of previous DVT and 43.46% showed an intact DVS.

In 1876, Le Dentú described 2 types of perforant veins: direct & indirect. The latter connect the Superficial System with a muscular vein of the leg and thence, through it, run into the Deep Venous System. They do not participate much in the disease, as during muscular contraction, the point of maximal reflux, with pressures ranging from 200 to 300 mm Hg provoke the total collapse of this muscular vein, so avoiding the reflux or buffering its magnitude.

On the other hand, direct perforants are those that directly connect the Superficial System with the deep principal or axial veins, which latter are submitted to lower pressures - 100 to 15° mmHg - during muscular contraction; they do not collapse totally but partially, in the middle. Direct perforants emerge from the lateral partly open edge and, due to the Venturi effect, tend to suction the blood towards the interior of the axial vein.

Direct perforants of the thigh and upper third of the calf run into larger cross-section veins like the Long Saphenous Vein or collaterals of similar caliber. Because of this and once perforant valvular insufficiency is established, the reflux is rapidly neutralized. On the other hand, the direct perforants in the lower part of the leg open up in a fine superficial vein, the Leonardo's vein which is usually of smaller caliber than them. This provokes the important dilatation of these perforants, and the impossibility of the superficial vein to neutralize the reflux, which progressively will reach the capillary bed and finally cause the trophic changes at skin level. We have observed that this situation takes place within what we called "Venous Buffer Circuit", formed by the Long Saphenous Vein to the front, Leonardo's vein to the rear and the LAA at bottom, which links both closing the "superficial circuit" at the submalleolous level. The superior, medial, inferior Cockett perforants and the fourth, submalleolar, which we had described, constitute the participating perforant system. At the deep level, the Posterior Tibial and Internal Plantar Vein complete the circuit. This Venous Buffer Circuit, which in the first stages is able to compensate the reflux by its superficial constituents; when global insufficiency ensues, the circuit fails to compensate the reflux thus creating permanent stasis and retrograde hypertension which transmits to the skin and underlying tissues originating the cutaneous lesions.

When the Deep Venous System participates in the insufficiency either caused by obstructive, essential, posthrombotic reflux, or a combination of these expressions, does not contribute to modify the magnitude of the reflux but its duration, possibly limited by the caliber of the insufficient perforant at the peak of the reflux.

Conversely, and determined by the destruction of the valves, the duration of the reflux is prolonged, as the reflowing blood amount increases and the size of the intervalvular compartments augments. This fact is responsible for the high incidence of venous ulcer.

Microscopically, the zone is severely affected; capillaries look elongated, dilated and tortuous mainly near insufficient direct perforants. Thrombosis of the capillary vessels interferes in skin nutrition process thus predisposing to ulceration. This phenomenon also compromises the initial lymphatics, which play a role in this pathophysiological process.